— MANGANESE & INFRASOUND — THE LINK TO MAD COW DISEASE

The rise in rates of BSE and vCJD is likely the result of a high manganese/ low copper mineral imbalance which compromises the mammalian brain's capacity to deal with incoming shock-bursts of infrasound.

Part 1 of 2

by Mark Purdey © 2002, 2003

High Barn Farm
Elworthy, Taunton, Somerset TA43PX
United Kingdom
Email: MadCowPurdey@aol.com
Website: http://www.markpurdey.com

An Underground, Ecodetective Journey

The White Sands Missile Range is an extensive spread of US military–controlled cactus country that spans the southernmost extremes of the San Andres Mountains in New Mexico. There is an eerie atmosphere to the place.

A Department of Natural Resources truck kicks up the dust across the droughty canyon, its engines reverberating in an agitated mode. It stops at the main entrance gates along the 12-foot-high perimeter fence. One of the wildlife officers gets out and walks to security, seemingly oblivious to the distant thump of a missile exploding across the range. He is clearly preoccupied with the more important task of slaughtering animals that have succumbed to this so-called "hyperinfectious" disease. The truck is soon on its way, loosing itself within the thousands of acres of parched-up military compound.

They've come to investigate yet another new eruption of chronic wasting disease (CWD)—the deer equivalent of "mad cow disease" (BSE). This outbreak is particularly significant, in that it represents the first cases of transmissible spongiform encephalopathy (TSE) disease recorded in a deer herd within the state of New Mexico. Furthermore, the affected herd has been confined behind the top-security perimeter fence for several decades.

This latest epidemiological aberration delivers a serious challenge to the viability of the conventional consensus on the origins of CWD. It has rumbled the cornerstones of institutionalised "expertise", questioning those who have plumped for the assumption that some unconventional "hyperinfectious" agent is spreading via body to body contact through the deer populations.

So how did the "infectious agent" jump the 500-mile gap between the CWD hotspot zone in Colorado and the CWD-free deer residing within the White Sands Missile Range? The "experts" were baffled. But, true to form, this latest challenge to the official theory was conveniently obfuscated into oblivion, outcast as some illusory mirage that just happened one day in the New Mexico desert.

But the answer is only evident to those who care to scratch a bit deeper than the dust. For they cannot help but notice some overt environmental features that exclusively predominate this unique location—factors which are invariably shared by every single TSE cluster location around the world.

Before the military came, White Sands was an industrial centre for the mining of manganese oxide and wulfenite ores (note that wulfenite contains the copper-chelating molybdenum metal). The museum-quality black crystals lay scattered across the top of the terrain. They twinkle out a kind of sombre resonance under the desert sun, emanating the haunted history of the place.

And since the military has occupied the range, the US authorities have been actively engaged in monitoring the unique intensity of infrasonic shock-bursts that are radiated by the explosions of their own missiles. The poor deer herd has played guinea pig to an unwitting experiment that has cracked the causal riddle of spongiform disease.

The BSE Debacle

Since 1986, the infamous novel neurodegenerative syndrome BSE (bovine spongiform encephalopathy) and vCJD (variant Creutzfeldt–Jakob disease) has insidiously blighted the heartbeat of British rural life. The disease has annihilated thousands of head of cattle and a growing number of young people, while creating a fierce battleground between nations, vested interests, political parties, farmers, victim support groups and consumers.

More recently, the shock-waves of the BSE debacle have ricocheted around the entire world.

But despite the severity of the "mad cow" legacy, little genuine attempt has been made to crack the causal riddle of the disease, thereby leaving us devoid of insight into measures that would best cure, control and, better still, prevent it.

This story shines a ray of light over the whole debacle. It charts my own ecodetective escapades and original field investigations which ran in tandem with the laboratory quest of Cambridge University biochemist Dr David Brown. These combined works have gone some way towards unearthing the truth underpinning the original causes of this grotesque disease.

Hard scientific evidence has been amassed which indicates that BSE and vCJD could both result from separate exposure of bovines and humans to the same package of toxic environmental factors—ferrimagnetic metals and low-frequency sonic shock—and not from the ingestion of infected bovine material. If such a polemical hypothesis continues to accumulate momentum, a radical upheaval of the status quo mindset can be expected.

But despite the conclusions of several field and laboratory studies providing strong support for the environmental hypothesis, the resulting publications have been dismissed outright by the UK government. Furthermore, contrary to the positive recommendations of the UK 2000 BSE Inquiry and EU Commission BSE reports in respect of funding research into this theory, the UK government's irrational rejection of grant proposals—including one submission aimed at developing a feasible cure for vCJD—continues to the present day.

I was struck by the fact that no cases of BSE had ever emerged in cows that had been born and raised on fully converted organic farms.

The Lone Voyager

My work first came to the fore after I successfully quashed the UK government's compulsory warble fly eradication regime in the High Court of London in 1984. This exempted my farming business from treating our dairy herd with a systemic organo-dithiophosphorus (OP) insecticide—a toxic chemical which, amongst myriad toxicological effects, can chelate copper and open up the blood–brain barrier, thereby disturbing the overall crucial balance of metals in the brain.

I was therefore not surprised to witness BSE rearing its ugly head in the UK cattle herd in 1985. In my opinion, it was a direct legacy of the UK government's compulsory warble fly eradication campaign—a 1982 measure that enforced the exclusive twice-annual high-concentration application of systemic-acting OP insecticide.

As considerably smaller outbreaks of BSE began to erupt across other European countries and later in Japan, my investigations revealed the voluntary usage of the same types of systemic insecticide in those countries—albeit at half the dose rates as applied in the UK. These European outbreaks seemed to follow an EU campaign, known as COST 811, which was aimed at purging the remaining bastions of warble infestation on the European mainland—in countries where outbreaks continued because their respective authorities had adopted a more laid-back, voluntary approach towards control of warbles.

In warble-free Japan, the BSE cases emerged in the specific herds which had imported breeding cattle from warble-infested North America; and so the Japanese took preventive measures by blanket-treating those herds with the same types of systemic OP that had been used in Europe. It should be pointed out that the USA wisely adopted a less toxic approach for dealing with their warbles, employing lower doses of non-systemic-acting insecticides—e.g., insecticides which were not designed to penetrate through the skin—while only treating the individual cattle that were warble infested.

I was a working dairy farmer with first-hand experience of BSE erupting in cattle that had been purchased into my organic farm. But I was struck by the fact that no cases of BSE had ever emerged in cows that had been born and raised on fully converted organic farms, despite those cattle having been permitted access to the feed that contained the incriminated meat and bone meal (MBM) ingredient as part of their 20% conventional feedstuff allowance decreed in the organic standards at that time.

From then on, I became deeply sceptical of the conventional consensus on the origins of BSE and its human equivalent, vCJD. There were just too many radical flaws blighting the hypothesis that bovine ingestion of micro doses of scrapie-contaminated MBM led to BSE. Equally flawed was the follow-up theory that human ingestion of BSE-contaminated beef caused vCJD.

The "hyperinfection hysterics" had based their hypothesis upon the notion that TSEs could be transmitted via injections of TSE-diseased brain tissues into unfortunate laboratory animals. Yet various other neurodegenerative diseases, such as familial Alzheimer's disease, have been transmitted in this way.

So why is nobody freaking out about Alzheimer's disease?

The Flaws in the Conventional Hypothesis

1. Thousands of tonnes of the BSE-incriminated meat and bone meal feed was exported as cattle feed during the 1970s/1980s/1990s to countries and regions that have remained BSE free to date—e.g., South Africa, Sweden, Eastern Europe, Middle East, India, Third World, etc.

2. Relaxation of the temperature standards and manufacturing techniques in the MBM rendering process in the UK was blamed for permitting the survival of the scrapie agent in sheep brain material, thereby enabling the "agent" to jump across into cattle, producing BSE. But none of these alterations was exclusive to the UK rendering plants. For instance, other scrapie-endemic places such as the USA and the Scandinavian countries had adopted the same continuous flow system of rendering five years before the UK did, yet these countries have remained BSE free. Furthermore, the pathogenic "infectious" capacity of the scrapie agent remains active after heating to temperatures in excess of 500 degrees—way above the 150-degree temperatures employed in the supposedly "safe" rendering processes operating in pre-BSE days.

3. Several abhorrent live-animal trials in the USA failed to induce BSE in cattle after feeding/injecting them with massive doses of scrapie-contaminated brain tissue.

4. Forty thousand–plus cows that were born after the UK's 1988 ban on MBM incorporation into cattle feed have still developed BSE.

5. Several countries such as Ireland, Portugal and France witnessed a greater number of BSE cases in cows born *after* their respective bans on MBM than in cows born before the bans.

6. In the UK there have been no cases of BSE in other TSE-susceptible ruminants such as goats and sheep, despite the customary inclusion of the same MBM protein source in their feeds.

7. Four of the original five kudu antelope that developed BSE at the London Zoo had not had any possible access to feeds containing MBM.

8. The UK government's former experimental farm at Liscombe on Exmoor was designed to raise suckler beef cattle on a pure grass/silage system, without resorting to feeding any MBM-containing concentrated feeds at all. Yet BSE struck down four animals on this holding.

9. The infamous mechanically retrieved meat products/baby foods blamed for causing vCJD in the UK were exported all over the world to countries where vCJD has not erupted to date. Likewise, the tradition of "skull splitting" in small rural butchers, which has been offered as an explanation for the growing number of vCJD clusters in rural areas, had been practised by the smaller butchers all over the UK.

10. BSE fails to fulfill "Koch's postulates"—the yardstick for gauging whether a given disease stems from infectious origins. For instance, more than 15% of cattle slaughtered for displaying the classic symptoms of BSE did not demonstrate the presence of the "causal" prions at post-mortem.

The Reductionist Mindset Takes a Firm Hold

Despite the myriad epidemiological flaws and millions of pounds worth of research failing to ascertain any association between the origin of these diseases and the scrapie agent, the whole propaganda myth that BSE was caused by scrapie became incorporated as "gospel" into the mainstream public/professional mentality.

But it is easy to see how the momentum of such a reductionist mindset took a hold. The media loved the theory because they could drum up a viral holocaust-horror scoop. The farming industry could get their beef sales back on the road by deluding consumers into believing that they had eliminated the causal agent. The vegetarian lobby found themselves landed with a powerful propaganda weapon on their plate, while the scientific institutions could carry on drawing generous funding for their hyperinfection witchhunt without the embarrassment of having to account for years of barking up the wrong tree. And the government could conveniently offload the blame onto the vagaries of some naturally occurring "nasty" for which no vested interest or official directive could ever be held accountable.

In the early days, the world of TSE research had been confined to the rather cranky ranks of backstreet institutions whose researchers seemed more preoccupied with advancing acidic debate over the nature of the "infectious" agent than with getting on with worthwhile research projects. It was these scientists who first fossilised the reductionist notion that TSEs stemmed from infectious origins.

But as soon as the positive evidence for the first case of BSE was back from the lab, a fast-expanding clique of "expert" microbiologists swooped in, hijacking all the research grants and rapidly laying claim to full ownership and academic rights over this new strain of TSE. They coined the classy name "prion disease", and ran a host of sharp-suited symposiums set in expensive five-star Floridian hotels, thousands of miles adrift from the English pasturelands—the hotbed of the real problem.

From then on, any investigations into the broader scientific perspectives surrounding TSEs were frozen out of the agendas of the funding bodies. Multidisciplinary research studies were forced to give way to research projects that conformed to the convergent assumption that the "prion" would encapsulate all of the answers to this problem. The journals were soon bursting apart with a monotonous dirge of articles that bleated out yet another variation on the stereotyped theme of the prion protein: prion protein genotypes, the biochemistry of the prion protein, along with a countless number of prion transmission live-animal studies that had been duplicated by virtually every institution involved with TSE research—for no useful scientific purpose.

Once it became clear that the various feedbans had failed to halt BSE in the UK (e.g., the 40,000+ BSE cases in cattle born after the 1988 feed ban), the incestuous clique of "expert" advisers was forced to come up with an ever-increasing array of implausible reasons for explaining the continuation of BSE. And following on from that advice, an equally inept package of control measures was implemented whenever and wherever TSE reared its ugly head around the world.

Their final farcical solution entailed a wholesale annihilation programme of both wild and domestic animal populations across specially designated TSE eradication zones. Despite the well-publicised history of total failure of these abhorrent control measures, this brave new wave of totalitarian overkill went unquestionably ahead—gobbling up millions of healthy mammalian lives and millions of dollars of public funds.

... nobody has offered a credible explanation as to how these abnormally shaped "prions" initially develop in the mammalian brain or the natural environment ...

Prion Origins: The Quest for the Primary Cause

While it is well established that the key pathological hallmark of the spongiform-diseased brain is represented by a malformed version of the prion protein, known as the prion, there is no actual evidence in support of the assumption that the protein portion of the prion represents the TSE infectious agent. Nor is there evidence that TSEs are infectious diseases that can be spread via body-to-body contact. And furthermore, nobody has offered a credible explanation as to how these abnormally shaped "prions" initially develop in the mammalian brain or the natural environment, nor explained how these prions are capable of inducing a kind of cascade of self-replication in the TSE-diseased brain.

I became interested in the possibility that the use of systemic OP warble fly insecticides may have triggered this protein malformation in some way, thereby serving as one of the primary causes of the modern BSE/vCJD strains of this disease. For these oil-based chemicals were designed to penetrate through the skin and metamorphose the internal environment of the cow into a poisonous medium so as to exterminate internal parasites. They had to

be poured along the head/backline of the cow, just millimetres from where the prion protein is manufactured in the cell lines of the spinal cord.

It was well recognised that OP insecticides exert their toxic effects in mammals by deforming the molecular shape of various nerve proteins like acetylcholinesterase, whereupon these malformed proteins cease to perform their proper function in the brain. But nobody had ever considered that a similar-style molecular interaction might occur between OPs and the prion protein. Since the prion protein has been shown to bond up with copper in the healthy brain, I felt that the ability of these dithio insecticides to lock up copper in the treated animal may play some role in deforming the prion protein.

After many abortive attempts to coerce the Establishment into running the correct laboratory test, I eventually raised funds from well-wishers and obtained personal loans to finance Dr Stephen Whatley of the Institute of Psychiatry in London to challenge brain cell cultures with the OP phosmet—the actual OP used at uniquely high doses on UK farms.

Amazingly, these trials demonstrated that the OP altered the cellular metabolism of prion protein in some of the ways observed in the early stages of spongiform disease—suggesting that phosmet exposure may render mammals more susceptible to the disease. Unfortunately, these experiments did not produce the key deformation of the prion protein that is seen in TSEs.

I returned to square one, assuming that OPs in combination with a further factor X could fulfill the final missing link in the causal jigsaw. Or perhaps OPs weren't involved at all!

A specific environmental source of manganese could be pinpointed in every TSE cluster zone that I had investigated to date.

The Cluster Buster

Caught in the midst of a minefield of multinational interests, medical spin doctors and political propagandists, I grew exhausted by the vortex of professionals who had successfully hijacked all UK scientific research into TSEs into a cul-de-sac.

Furthermore, I'd found myself frozen out into the cold, operating much like an underground scientist, tramping a covert journey around the rustic outbacks of the UK's farmlands to nail down the true cause of BSE.

So I expanded my horizons and embarked upon a refreshing ecodetective trek to analyse the unique environments around the world where traditional TSEs had erupted as high-incidence clusters for many years. By scanning the overall essence of each cluster location, I attempted to pinpoint the common causal factors—the aetiological needles in the causal haystack.

Against a backdrop of flamboyant and sometimes threatening scientific scenery, I embarked on a lone journey to the ends of the Earth, sampling exotic corners of Colorado, Iceland, Slovakia, Calabria, Sardinia, Japan, etc.—areas where an assortment of animals and humans had demonstrated a high incidence rate of TSE. My analytical results displayed abnormally high levels of the metal manganese and rock-bottom levels of copper, selenium and zinc in common in all of these food chains. Levels of manganese were normal in adjoining disease-free areas.

I was also fascinated to discover that these TSE-affected populations lived in areas that were enduring front-line exposure to intensive shock-bursts of low-frequency infrasound—from military and quarry explosions, volcanic and earthquake activity, low-flying supersonic jets, *Concorde* overflights, etc.

A specific environmental source of manganese could be pinpointed in every TSE cluster zone that I had investigated to date, where each TSE-affected ecosystem could be directly connected to the atmospheric fallout of some naturally occurring or industrial source of combusted manganese oxide, e.g., stemming from volcanic activity, acid rain, steel/glass/ceramic/dye/munitions factories, lead-free petrol refineries, the take-off airspace beyond airports, etc. In this respect, it should be borne in mind that atmospheric manganese, much like silver and aluminium, can be absorbed directly into the brain via the nasal-olfactory inhalatory route of intake. Perhaps this highly efficient mode of uptake enables sufficient concentrations of manganese to accumulate in the brain and initiate TSE?

Furthermore, many of the mammalian populations involved in the outbreaks of both traditional and new-strain TSE could be linked to the consumption of high-concentration manganese supplements for bone growth, etc. For instance, the clusters of chronic wasting disease in deer could all be linked to the areas where a "spiced up" manganese mineral lick had been put down by the deer hunters for addicting deer to their shooting territories. The manganese is added for promoting antler growth. In fact, *all* species connected with TSEs—deer, cows, goats, mink, sheep, zoo animals, cats, humans, etc.—are given manganese supplements in their feed.

Disturbingly, manganese is also added to milk substitute powders for calves (and human infants) at levels up to 1,000 times those found in normal cow's milk. This practice has been widespread in all countries affected by BSE to date.

Excessive intake of dietary manganese poses a great risk for the immature mammal, since the regulatory mechanisms of the blood–brain barrier are underdeveloped at this stage, thereby permitting an excessive uptake of manganese and other metals into the brain.

The addition of manganese to substitute milk powders explains why European dairy cattle—which were invariably reared on this powder—suffered such high incidence rates of BSE in relation to the negligible rates in beef suckler and organically reared cattle—which were invariably reared on natural cow's milk.

Every Storm Cloud Has A Silver Lining

A few other TSE cluster hotspots had demonstrated the same low-copper connection but had measured abnormally high levels of other potentially toxic transition metals such as silver, platinum and lithium as well as manganese. Much like manganese, these metals will also readily substitute at copper bonds on prion proteins.

Some of these TSE environments were silver-mining areas where local ecosystems were naturally high in silver, while others

were centred around ski resorts, reservoirs, airport flight paths, coastal districts, and sites where extensive aerial spraying of weather-modifying silver iodide "cloud seeding" chemicals had been used for inducing rainfall/snowfall and dispersing cloud/fog.

Metals like silver and platinum are also used as key ingredients in dental amalgam fillings, surgical depth electrodes/instruments, etc., perhaps explaining why dental treatments and surgical operations/electrode implantations are considered to be high-risk prerequisites for triggering CJD.

Manganese Breaketh Man

The recent surge in the global rates of TSEs (and other neurodegenerative diseases) seems to have run in tandem with the increased incorporation of high-concentration manganese oxide additives into the bovine, human, pet and zoo animal food chains—via a multitude of applications such as free-access mineral licks, supplement tablets, fertiliser and fungicide sprays, paints, petrol additives, etc., or via increased consumption of trendy food products, such as soya, which naturally bioaccumulate high levels of this metal from the soil.

Given the fact that long-term manganese exposure is well known to induce a wide array of progressive neuropsychiatric degenerative disorders—dubbed the "manganese madness" syndromes—in factory welders and mine workers, etc., the idea that manganese could perform a front-line role in the pathogenesis of TSEs is not out the way.

In fact, manganese toxicity can manifest itself in many forms. When I visited areas in the South Pacific, like the island of Guam, where a raft of neurodegenerative conditions involving Alzheimer's, Parkinson's and motor neurone diseases used to run at a fiftyfold higher incidence rate than elsewhere across the world, I noticed that manganese mining used to be widely practiced in all of these areas.

And then I arrived on Groote Eylandt, a once-upon-a-time enchanted tropical island in the Gulf of Carpentaria in northern Australia, whose history has witnessed a bizarre degree of "heaven and hell". Not only do the island's soils play host to the mother of all manganese concentrations, but its flamboyant rainforest ecosystems have supported ideal hunter-gatherer grounds for some of the most pure bred, nomadic Aboriginal clans alive in Australia today. But, as I was soon to learn, the tropical charms of a Groote Eylandt of "pick-your-own" coconuts and turquoise seas can be deceptive to the uninformed outsider.

I found that 3% of an indigenous Aboriginal clan residing around one of the largest open-cast manganese mines in the world had recently been struck down by a bizarre ataxic syndrome—what turned out to be caused by a high manganese/low magnesium–induced mutation in early life.

Former fit and healthy Aboriginal warriors were finding themselves progressively transfigured into debilitated and wasted neurodegenerative wrecks—more akin to stick insects trying to cross ice. Furthermore, the levels of unrestrained aggression and murder in this community had reached crisis proportions, where grotesque "Hieronymus Bosch–style" brawls erupt on a weekly basis.

In this respect, the link between manganese and aggression has been well established, where manganese has been recorded at elevated levels in the bodies of those who have been executed on death row for violent crimes. Professor Louis Gottschalk, the man who carried out these studies, reckons that "manganese levels serve as a marker for violence".

But, true to form, the official cause of this "drunken walking" syndrome has been conveniently scapegoated onto a mutation caused by faulty Aboriginal "seed".

To challenge this dogmatic assumption, I travelled several thousand miles to another isolated cluster of this same ataxic condition that exists amongst indigenous folk living on the islands of Flores and São Miguel in the Azores archipelago. After drawing a range of environmental samples, I unearthed the same abnormal mineral ratio that is found on Groote. In fact, the levels of manganese were so high in the black volcanic soils of these islands that the mining prospectors had considered it lucrative to mine the metal from the local Azorean seabed.

Given the high intensity of various neurodegenerative diseases that have invariably erupted around all of the manganese hotspot regions, I was beginning to wonder whether the increased exportation of manganese dioxide ores into the steel, glass, dye, lead-free fuel, paint and mineral feed supplement industries across the developed world had somehow seeded the "mad cow" madness in the deer, sheep, cats, zoo animals, cows and teenagers.

These experiments represented the first time that malformed prion protein had been created experimentally as a *de novo* transformation.

Laboratory Studies Support the Theory

My observations of high manganese/low copper in TSE cluster areas led to my connecting with the pioneering laboratory investigations of Dr David Brown at Cambridge University in the UK. He is a widely published biochemical expert who had pursued groundbreaking studies which unearthed interesting new facets of the elusive prion protein.

Dr Brown had demonstrated that the prion protein bonds to copper in the normal healthy brain. In this respect, his lab studies were complementary to my field studies in that they provided the other half of the necessary groundwork upon which I formulated an holistic hypothesis on the origins of these diseases.

I published a paper proposing that manganese could substitute itself at the vacant copper site on the prion protein, whereby this aberrant substitution by a foreign metal co-partner could induce the all-important deformation of the protein that is considered to be crucial to the development of TSE. In this respect, any TSE-susceptible mammals who were dependent upon these high manganese/low copper TSE cluster food chains would be at risk of developing TSE.

So David Brown ran the necessary cell culture experiments where he introduced manganese into copper-depleted prion protein cell cultures. Amazingly, his experiments produced the key deformation of the prion protein which the earlier tests using OPs had failed to create. These experiments represented the first time that malformed prion protein had been created experimentally as a *de novo* transformation.

Furthermore, follow-up trials by the USA's Prion Surveillance Center at Case Western University, Cleveland, looked at post-mortem tissue samples taken from CJD brains. Their analyses revealed the same abnormal mineral ratio as identified in the TSE

cluster environments—a tenfold higher level of manganese and a 50%-reduced level of copper in relation to control brains. Interestingly, the manganese was bonded to the abnormal prions in these CJD tissues.

It seemed that the basic cornerstone of the environmental theory was beginning to establish itself, and an overall picture of the pathogenesis of disease was panning out: that a high manganese/low copper imbalance somehow compromised the brain's ability to deal with acute shock-bursts of sound and light—the other common characteristics of TSE ecosystems.

My investigations indicated that the traditional forms of TSE—which tend to surface in elderly mammals—represent a less intensive mode of exposure to naturally occurring environmental influences which bring about a high manganese/low copper ratio in the mammalian brain.

Whereas, the more aggressive "rapid attack" modern strains of TSE—BSE, vCJD—can be explained by the current trend of increased mammalian exposure to a modern cocktail of man-made pollutants involving manganese compounds and copper-chelating chemicals (e.g., the warble fly and headlice OP insecticides). These penetrate into the central nerves and give rise to the more virulent, accelerated version of TSE, where full-blown symptoms of TSE erupt in much younger mammals than normal.

But unfortunately, the abnormally shaped prions, which Brown's high manganese/low copper experiments had created, failed to demonstrate the bizarre "infectious" multireplicating property that has been associated with the fully fledged prion in the TSE-diseased brain.

Could the further infrasonic shock factor that I had identified in the TSE cluster regions fulfill this final missing piece in the causal jigsaw? Perhaps some "shock-induced" quantum facet of the manganese atom may provide that final clue.

One of the interesting properties of manganese is that it can absorb the energy of sound.

Quantum Capacities: Final Key to the Causal Jigsaw?

Modern health authorities could learn a lesson from the alchemists of the Byzantine era who regarded manganese as the black magic metal, whereby the quantum capacity of manganese to absorb light and sound energy can induce a lethal "Jekyll and Hyde"–style conversion of this metal from innocuous to toxic form.

Interestingly, the initial pathological damage of TSE is manifested within the diseased mammal's retina, eyelid, skin, auditory and optic nerve endings—areas that perform a front-line role in neutralising the deleterious effects of incoming sound and light from the external environment.

Furthermore, the normal copper-bound prion protein is exclusively manufactured in these same tissues: the retina, spleen, lymphatics, tonsils, gut membranes, growth/repair cells, myocardium, pineal gland, visual cortex, pituitary gland, sympathetic neurones, etc.—tissues whose metabolism is regulated by the circadian daylight/darkness rhythm.

Laboratory animals that have been cruelly subjected to GM prion protein "knock-out" experiments have demonstrated that they are no longer able to regulate their sleep, sex and immune cycles—a sick way of demonstrating the role of the prion protein in mediating the circadian rhythm.

Copper Prions as the Conductors and Manganese Prions as the Blockers of Electromagnetic Energy Flow

The simple fact that copper is employed in electrical cables for conduction of electrical energy, whereas manganese is employed in batteries for storing electrical energy, offers a feasible explanation for both the function/dysfunction of the prion protein as well as the cause of TSEs.

Perhaps the copper element of the normal healthy prion protein plays a role in the conduction of electromagnetic energy along the circadian/auditory pathways of magnetic superexchange, where a linear chain of paramagnetic copper atoms (bonded to prion proteins) provides a "metal to metal to metal…" motorway which distributes the energy of light and sound around the body for energising the cycles of sleep, sex, behaviour, heartbeat, cell growth/repair and immune response. Whereas the abnormal manganese prion serves to block the pathways of electromagnetic superexchange by storing that electromagnetic energy to a level where the critical threshold of "flashpoint" is exceeded, thereby detonating neuropathogenic cluster bombs of free-radical chain reactions that progressively degenerate the circadian pathways of the brain—and cause TSE. In this respect, the blockage of the electromagnetic energy flow by such manganese replacement of copper can precisely account for the clinical and pathological profiles of the TSE disease process.

It could be said that the discovery of the function of the prion protein may turn out to give further scientific substance to the existence of the electromagnetic meridians recognised by Chinese medicine—where the healthy copper prion performs a regulatory role in maintaining the electrohomoeostasis along the acupuncture meridians and nodes.

While David Brown's manganese experiments had created the infamous deformed prion protein, they had failed to produce the all-important pathogenic "infectious" prion. Furthermore, manganese is a paramagnetic metal (i.e., a metal that can be temporarily magnetised), just like copper, so to some extent you would expect the manganese to substitute effectively for the electromagnetic conduction function of copper without too much negative repercussion. Perhaps some further modification of the manganese atom initiates a pathogenic capacity that is capable of kicking off the full-blown pathogenesis of TSE.

An Infrasound-induced Metamagnetic Transformation of the Manganese Atom?

One of the interesting properties of manganese is that it can absorb the energy of sound—well illustrated by its use (as with chromium, etc.) in some audiotape materials for storing a memory-bank of sound recordings in magnetic form. But manganese can only couple up with phonon energy when it occurs in its trivalent octahedral crystalline form.

So if atmospheres or soils containing manganese 3+ atoms are exposed to high intensities of infrasonic shock energy, these low-frequency vibrations are sufficient to metamorphose the actual atomic structure of the manganese atom itself, creating a kind of "Jekyll and Hyde"–like transformation of manganese from its normal paramagnetic form to rogue ferrimagnetic form, i.e., from a temporary to a permanent magnetisable form.

Once an ecosystem has been infrasonically adulterated, any manganese 3+ minerals that have been exposed to the full force of infrasonic shock—from military explosions, supersonic overflights, volcanic eruptions and tectonic activity, etc.—would leave a permanent legacy of rogue ferrimagnetic manganese atoms that are free to infiltrate the food chain, thereby contaminating any mammalian population that is dependent upon the local ecosystem in the years to come. If the adulterated food chains are simultaneously short of copper, then the mammalian population is put at high risk of developing TSE.

Map 1:
Geographical distribution of vCJD cases at onset of symptoms at 2001.
(Source: CJD Surveillance Unit, Edinburgh, UK)

Map 2:
Areas in the UK where manganese deficiency can occur in crops, so where spraying of manganese fertiliser occurs.

TSE Clusters Correlate to the Epicentres of Infrasound

The overriding presence of this package of environmental prerequisites has been identified in every TSE cluster zone investigated to date. Besides the White Sands Missile Range, another clearcut example of the presence of infrasound involved an intensive outbreak of scrapie after sheep had been moved onto a block of copper-deficient common land at Ashoro on the Hokkaido Peninsula. This land was previously occupied by the Japanese military during World War II, where intensive explosions were a daily occurrence.

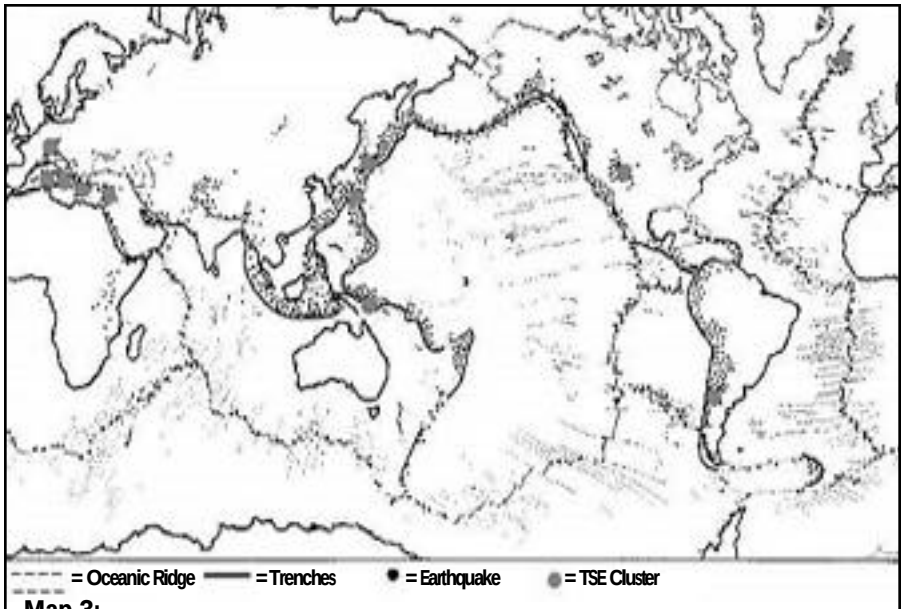
And then there is the well-renowned, long-established CWD hotspot zone that runs along the copper-deficient Front Range foothills region of Fort Collins in Colorado. This area used to play host to the supersonic test flights of the military jets from nearby Warren Air Force Base during the late 1960s/early 1970s. When the complaints of Fort Collins residents reached screaming point, the military was forced to move the flights to the plains in the northeast—and CWD outbreaks have followed likewise.

Furthermore, my investigations have identified that the new wave of CJD, BSE and scrapie clusters that have erupted across southern Italy, Sardinia and Sicily since the mid-1990s are invariably located in areas beneath the flight paths radiating out of the intensive number of NATO bases recently sited within this strategically important military position in the Mediterranean.

As a rule, the traditional forms of TSE result from exposures to the naturally occurring infrasonic shock waves that radiate from geotectonic plate rift lines (earthquakes, volcanic activity, etc.), whereas the new-strain TSEs result from the more intensive low-frequency shock-bursts radiating from supersonic military and *Concorde* aircraft. The emergence of the respective types of TSE clusters along these rift lines and flight path lines substantiates this idea well.

Perhaps it is no surprise that 99% of the total cases of vCJD and BSE to date are contained within Britain and France, both countries having exclusively developed the *Concorde* aircraft, thereby unwittingly exposing themselves to the most intensive source of artificial infrasound unleashed to date.

In this respect, all of the clusters of new variant CJD that have erupted in rural regions in the UK lie beneath the routine and charter flight paths of *Concorde* and low-flying military jets. The afterburner turbofans employed by these supersonic aeroplanes radiate such a high intensity of low-frequency infrasound that a



Map 3:
Correlation between locations of long-term cluster of sporadic/familial TSEs and areas of seismic activity along the major global tectonic plate margins.

100-km-wide carpet of acoustic shock-waves is left in their wake, whether the aircraft are flying subsonically or supersonically. Racing pigeons which have flown into this shock carpet have failed to return home, having permanently lost their sense of magnetic orientation.

Some interesting epidemiological data on BSE clusters was first presented to the BSE Inquiry by Dr Richard Morris, an epidemiologist from New Zealand. He showed that the most intensive clusters of BSE had always appeared on the extreme tips of the copper-deficient west coast peninsulas in the UK. How could this pattern relate to MBM feeding?

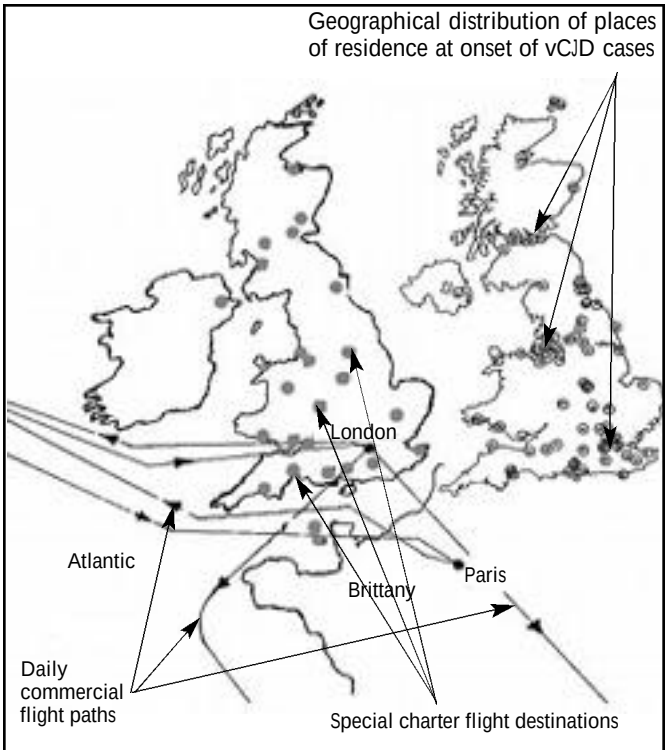
Intriguingly, the well-used west coast route for supersonic military and *Concorde* aircraft runs precisely over these BSE hotspot locations.

It is perhaps no surprise that the infrasonic environs of Staten Island and Long Island in the USA—both under the flight paths of John F. Kennedy Airport where *Concorde* and other aircraft take off and land—have demonstrated the highest incidence cluster of traditional CJD in the USA.

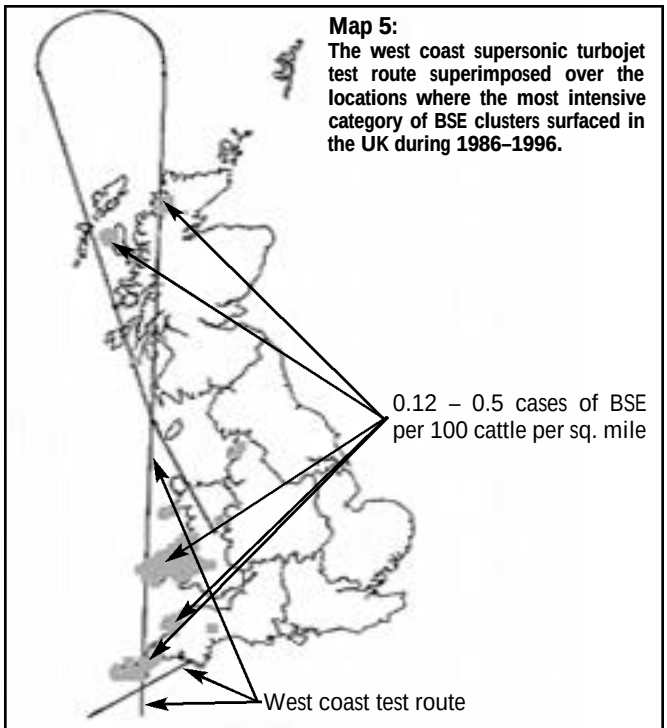
The pattern of emergence of both traditional and new variant TSE clusters in rural/coastal areas, as opposed to urban areas, substantiates well the hypothesis of an environmental origin. For rural/coastal ecosystems have become increasingly exposed to a toxic combination of manganese-based fertilisers/fungicides, copper-chelating pesticide sprays as well as the infrasound resulting from the overflights of low-flying turbojet aircraft that have been prohibited over urban areas of high population density.

Furthermore, this geographical pattern of TSE emergence helps to dispel the myth that CJD arises from ingestion of TSE-affected animal products, since meat products are consumed equally by urban and rural populations alike.

Continued next issue



Map 4:
Flight paths of *Concorde* supersonic aircraft to and from London and Paris airports and to Barcelona and the Middle East.



Map 5:
The west coast supersonic turbojet test route superimposed over the locations where the most intensive category of BSE clusters surfaced in the UK during 1986–1996.

(Source: Prof. R. S. Morris, Evidence to the BSE Inquiry, 14 Oct 1999)

Bibliography

NB: For additional references, please refer to the "Cattle Practice" article on the scientific pages of Mark Purdey's website, http://www.markpurdey.com.

• Purdey M. Ecosystems supporting clusters of sporadic TSEs demonstrate excesses of the radical-generating divalent cation manganese and deficiencies of antioxidant co-factors Cu, Se, Fe, Zn. Does a foreign cation substitution at Prp's Cu domain initiate TSE? *Medical Hypotheses* 2000 54(2):278-306.

• Purdey M. Does an ultraviolet photo-oxidisation of the manganese-loaded/copper-depleted prion protein in the retina initiate the pathogenesis of TSE? *Medical Hypotheses* 2001 57(1):29-45.

• Purdey M. The Mn-loaded/Cu-depleted bovine brain fails to neutralise incoming shock-bursts of low-frequency infrasound. The origins of BSE? *Cattle Practice* October 2002 10(4):311-325.

• Brown D, Hafiz F, Glassmith L, et al. Consequences of manganese replacement of copper for prion protein function and proteinase resistance. *EMBO J* 2000 19(6):1180-1186.

• Gordon I, Abdulla EM, Campbell IC, Whatley SA. Phosmet induces up-regulation of surface levels of cellular prion protein. *Neuroreport* 1998 9(7):1391-1395.

• Wong BS, Chen SG, Colucci M, Xie Z, Pan T, Liu T, Sy MS, Gambetti P, Brown DR. Aberrant metal binding by prion protein in human prion disease. *J Neurochem* 2001 78:1400-1408.

About the Author:

Mark Purdey is a traditional mixed family farmer in the UK who successfully defeated the UK government's compulsory insecticide treatment order in the High Court of London in 1984. He writes, lectures and broadcasts on environmental health issues, whilst pioneering global ecodetective investigations into the causes of brain disorders, such as BSE. Mark's analytical studies have identified some "common toxic denominators" involved in the cause of BSE, and his published hypothesis is currently gaining support from studies conducted at US, Japanese and European universities.

The full text of Mark's article can be found on his website, http://www.markpurdey.com. It was originally published as "Educating Rida"—"Rida" being the Icelandic term for transmissible spongiform disease (TSE).